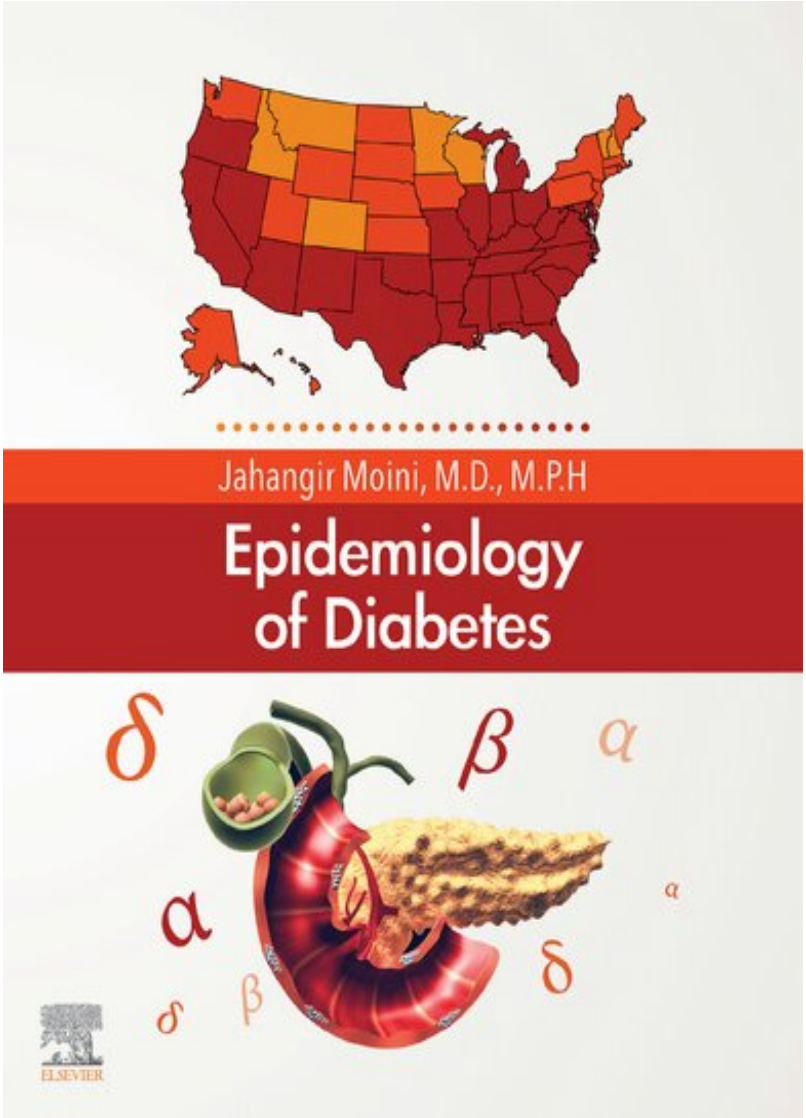
Epidemiology of Diabetes 1st Edition M.D. Moini pdf download

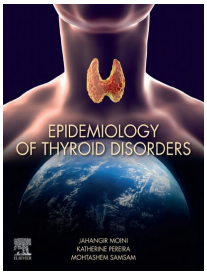
https://ebookmass.com/product/epidemiology-of-diabetes-1st-edition-m-d-moini/



Explore and download more ebooks at ebookmass.com

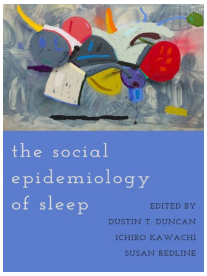
We have selected some products that you may be interested in
Click the link to download now or visit ebookmass.com
for more options!.

Epidemiology of Thyroid Disorders 1st Edition M.D. Moini



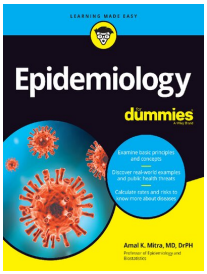
https://ebookmass.com/product/epidemiology-of-thyroid-disorders-1st-edition-m-d-moini/

The Social Epidemiology of Sleep 1st Edition Dustin T. Duncan



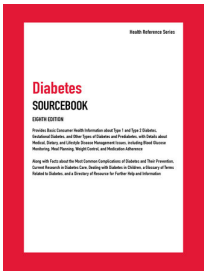
https://ebookmass.com/product/the-social-epidemiology-of-sleep-1st-edition-dustin-t-duncan/

Epidemiology For Dummies 1st Edition Amal K. Mitra



https://ebookmass.com/product/epidemiology-for-dummies-1st-edition-amal-k-mitra/

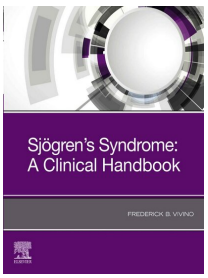
Diabetes Sourcebook, 8th Edition Kevin Hayes



https://ebookmass.com/product/diabetes-sourcebook-8th-edition-kevin-hayes/

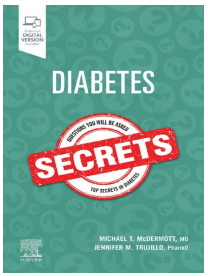
Sjogren's Syndrome: A Clinical Handbook 1st Edition M.D. Vivino

https://ebookmass.com/product/sjogrens-syndrome-a-clinical-handbook-1st-edition-m-d-vivino/



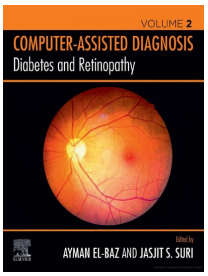
Diabetes Secrets 1st Edition Michael T. Mcdermott Md Author

https://ebookmass.com/product/diabetes-secrets-1st-edition-michael-t-mcdermott-md-author/



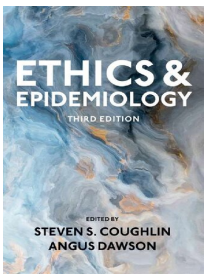
Diabetes and Retinopathy 1st Edition Ayman S. El-Baz

https://ebookmass.com/product/diabetes-and-retinopathy-1st-edition-ayman-s-el-baz/



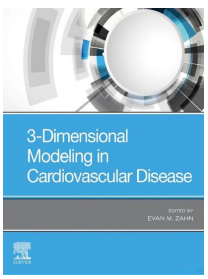
Ethics and Epidemiology 3rd Edition Steven S. Coughlin And Angus Dawson

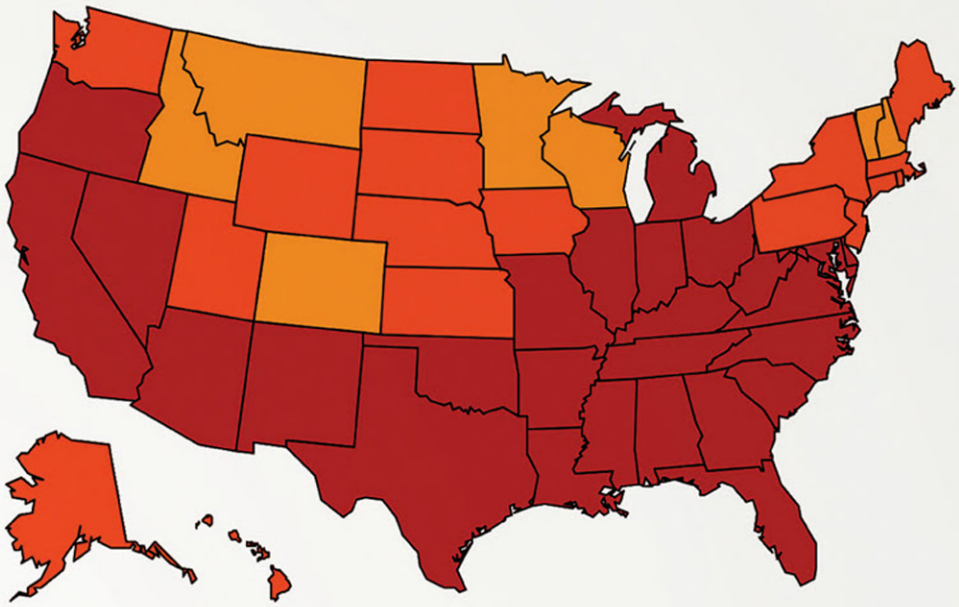
https://ebookmass.com/product/ethics-and-epidemiology-3rd-edition-steven-s-coughlin-and-angus-dawson/



3-Dimensional Modeling in Cardiovascular Disease 1st Edition M.D. Zahn

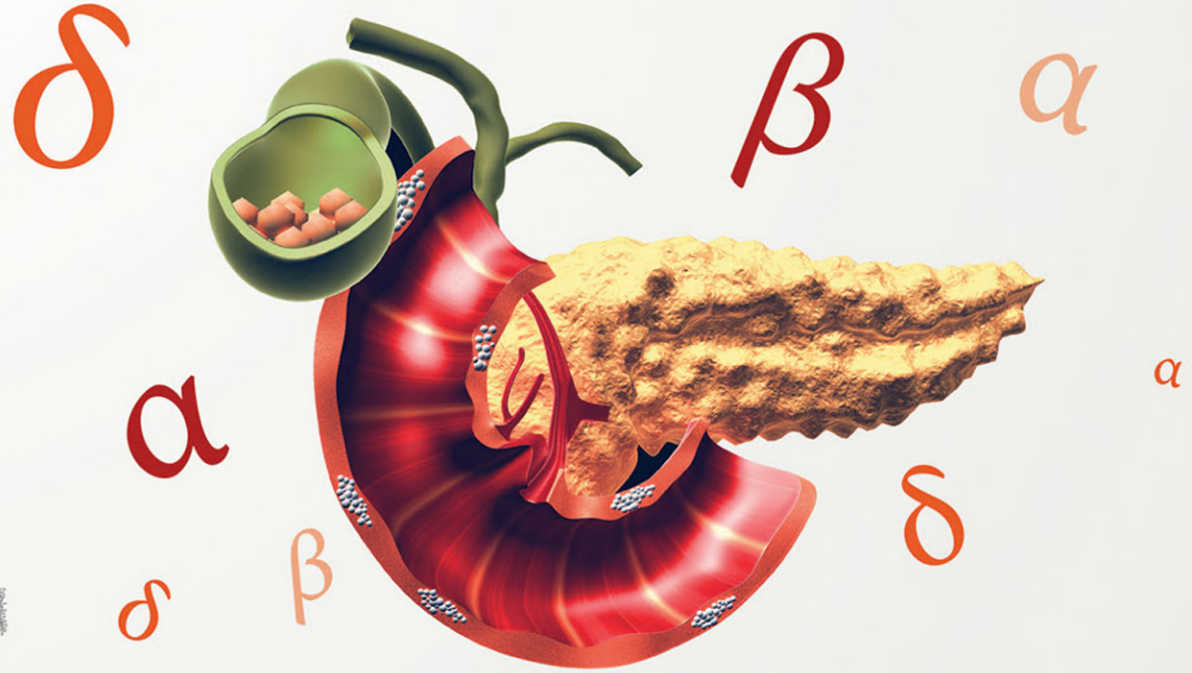
https://ebookmass.com/product/3-dimensional-modeling-in-cardiovascular-disease-1st-edition-m-d-zahn/





Jahangir Moini, M.D., M.P.H

Epidemiology of Diabetes



ELSEVIER

Epidemiology of Diabetes

Epidemiology of Diabetes

Jahangir Moini, MD, MPH
Professor of Science and Health
Eastern Florida State College, Palm Bay, FL, United States

Elsevier
Radarweg 29, PO Box 211, 1000 AE Amsterdam, Netherlands
The Boulevard, Langford Lane, Kidlington, Oxford OX5 1GB, United Kingdom
50 Hampshire Street, 5th Floor, Cambridge, MA 02139, United States

Copyright © 2019 Elsevier Inc. All rights reserved.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Details on how to seek permission, further information about the Publisher's permissions policies and our arrangements with organizations such as the Copyright Clearance Center and the Copyright Licensing Agency, can be found at our website: http://www.elsevier.com/permissions.

This book and the individual contributions contained in it are protected under copyright by the Publisher (other than as may be noted herein).

Notices

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices, or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds, or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

To the fullest extent of the law, neither the Publisher nor the authors, contributors, or editors, assume any liability for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

British Library Cataloguing-in-Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging-in-Publication Data
A catalog record for this book is available from the Library of Congress

ISBN: 978-0-12-816864-6

For Information on all Elsevier publications
visit our website at https://www.elsevier.com/books-and-journals

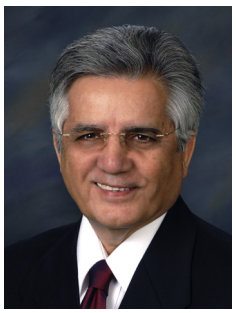
Publisher: Stacy Masucci
Acquisition Editor: Tari K. Broderick
Editorial Project Manager: Samuel Young
Production Project Manager: Sreejith Viswanathan
Cover Designer: Christian J. Bilbow

Typeset by MPS Limited, Chennai, India

Dedication

**This book is dedicated to my loving and amazing wife Hengameh,
and to my daughters Mahkameh and Morvarid, and also
to my two beautiful granddaughters, Laila and Anabelle.**

About the Author



Dr. Jahangir Moini was Assistant Professor for the Department of Epidemiology and Preventive Medicine, Medical School, Tehran University for 9 years.

For 18 years, he was the Director of Epidemiology for the Brevard County Health Department.

For 15 years, he was the Director of Allied Health for Everest University. He was also a Professor of Allied Health at Everest for a total of 24 years. For 6 years, he been a Professor of Science and Health at Eastern Florida State College.

He has been actively teaching for 39 years, and has been an international author of 35 books for 20 years.

Preface

Today, diabetes mellitus is one of the most common chronic diseases, with more than 30 million people affected in the United States. The disease has been globally relevant since the early 1980s. Modern lifestyles, such as increased caloric intake and decreased physical activity, are huge contributors to diabetes. Obesity is another factor that has increased globally, especially in the United States, with 70% of people being overweight or obese. The pandemic of diabetes is linked to a large variety of factors. These include genetics, nutrition, automation, urbanization, food costs, foods that are higher in calories and fat, and a more sedentary lifestyle.

There are a variety of complications created by diabetes, which include hypertension, stroke, heart attack, blindness, neuropathy, nephropathy, amputations, and death. The total estimated financial cost of diabetes mellitus in the United States, in 2017 alone, was $327 billion. This includes costs of treatment, disability, and premature death. The improvement of the outcomes of diabetes requires better testing, earlier diagnosis, patient education, more access to care, and improved quality of care.

This book includes information about the history of diabetes, the structure and functions of the pancreas, classifications of insulin, the role of insulin in metabolism, hypoglycemia, hyperglycemia, various types of diabetes, the prevalence and epidemic of diabetes, the effect of diet upon obesity, health impacts of diabetes, and diabetic ketoacidosis. It also contains up-to-date information about the diagnosis and treatment of diabetes mellitus, along with preventative measures.

Acknowledgments

The author appreciates the contributions of everyone who assisted in the creation of this book, including Stacy Masucci, Tari K. Broderick, Samuel Young, Sreejith Viswanathan, and Christian J. Bilbow. Also, the author would like to thank Dr. Morvarid Moini for contributing artwork, and Greg Vadimsky, who was the author's assistant.

Chapter 1

Introduction and History of Diabetes Mellitus

Diabetes is very common in the United States, with more than 30 million Americans having the disease. This makes up 9.4% of the country's population. Diabetes is the seventh leading cause of death in the United States. The condition known as diabetes mellitus is characterized by *high fasting blood glucose*. This is tested by taking a blood sample following an overnight period in which the patient has not consumed any types of food.

There are two primary forms of diabetes, which are known as *diabetes type 1* and *diabetes type 2*. In type 1, the beta (ß) cells of the pancreas can no longer secrete *insulin*. As a result, blood glucose levels become elevated to a condition called *hyperglycemia*. Almost 75% of type 1 diabetes cases are diagnosed in people under the age of 18 years. Type 2 diabetes occurs when the beta cells of the pancreas can no longer allow glucose to enter in order to produce energy. Studies have shown that the risks of developing type 2 diabetes are higher in people who have a family history, are inactive, and are overweight or obese.

Diabetes has become much more widely understood since the early 1980s, though the disease has existed for much longer. The perception is that a "newer" disease surrounds the components of the modern diet and lifestyle. Diabetes mellitus is related to less-than-adequate physical activity, increased caloric intake, and the resulting factor of obesity being much more common than in previous times. This situation occurs all over the world. Written records about diabetes have existed for thousands of years, in many different countries.

HISTORY

The first recorded record of diabetes mellitus comes from India, during the 5th century BC. Descriptions of the disease included the terms *emaciation, excessive urination with a sweet odor*, and *thirst*. In ancient Egypt (approximately 460 and 1550 BC), the disease was described as including *excessive urination*, but there was no description of a "sweet odor." In China (475−221 BC and AD 229) diabetes was described similarly to the Indian description, and it is believed that the people who wrote the symptoms down were describing patients with type 2 diabetes. They linked it with the following risk factors: *large quantities of calories*, *eating of cereal, rice, and sweets*, and as being more common in *rich people*—meaning that they could better afford these kinds of foods.

The actual term "diabetes" is credited to Demetrius of Apameia (approximately the 1st or 2nd century BC). The term is derived from the Greek language, originally meaning "to siphon" or "to empty." The first clinical description of diabetes came from Aretaeus of Cappadocia (approximately AD 81−138). In his book called *Therapeutics of Chronic Diseases*, he even described a "cure" for the disease.

Both Aretaeus and Galen of Pergamum (approximately AD 129−200) believed that the kidneys were the source of urine, and that diabetes was a rare form of kidney disease. Galen (Fig. 1.1) experimented on dogs to substantiate his findings. Both of them did not use any term describing the "sweetness" of urine. It is possible that they were actually describing *diabetes insipidus*, which is caused by lack of antidiuretic hormone or antidiuretic hormone, and not diabetes mellitus.

For the next 500 years, Greek physicians were very approving of Galen's work concerning diabetes. They added new medical writings focused mostly on treatments for the condition, which included bloodletting, medicinal herbs, and even various types of wine. The first diagnostic test for diagnosing diabetes was by actually tasting urine. The sweet taste was written about by the Asians, Egyptians, and Indians. In AD 229, Chang Chung-Ching commented that *the urine was so sweet that dogs liked it*. In the 5th century, medical texts from the Hindu culture described the urine as being like sugarcane or sweet honey. In AD 630, Theodore Protospatharios recommended that diagnosis of diabetes should include examination of the heated residue of urine.

Epidemiology of Diabetes. DOI: https://doi.org/10.1016/B978-0-12-816864-6.00001-8
© 2019 Elsevier Inc. All rights reserved.

FIGURE 1.1 Drawing of Galen.

Between the years 980 and 1638, Avicenna (an Iranian physician), Paracelsus (a Swiss physician), and Morgagni (an Italian anatomist) also described the sweet taste of diabetic urine. Avicenna is shown in Fig. 1.2. Paracelsus concluded that the condition was caused by salt deposits within the kidneys. This led to excessive thirst or *polydipsia* and excessive urine output or *polyuria*. His conclusion was derived from observing an unknown white-colored substance that remained after diabetic urine was evaporated.

In 1674, Thomas Willis, an English physician, tasted diabetic urine and named the disease as "diabetes mellitus." The term *mellitus* actually means "honey sweet." In his book entitled *Pharmaceutice Rationalis*, Willis differentiated diabetes mellitus from diabetes insipidus. By 1776, Robert Wyatt and Mathew Dobson had proven that the sweetness in diabetic urine was accompanied by sugar in the blood of these patients.

By the 19th and early 20th centuries, the ability to quantitatively test for elevated sugar in the urine or *glycosuria* and elevated sugar in the blood or *hyperglycemia* was developed. Research was conducted on the digestive system, liver, and pancreas by American, French, German, and Italian scientists. Apollinaire Bouchardat was a diabetologist who, in the 1800s, identified the disappearance of glycosuria in Parisian diabetics, during the Franco-Prussian War, as a result of food rationing. Bouchardat treated diabetics with low carbohydrate diets and fasting. He documented that exercise and weight reduction improved their metabolism. He also developed the first steps in educating patients about their own responsibility to control diet and exercise, to prevent diabetes. In 1921, insulin was discovered in Canada by Frederick Banting, Charles Best, James Collip, and John James Rickard Macleod. This discovery was significant in that it paved the way for type 1 diabetes to become survivable, instead of a rare, fatal disease.

ORIGINS

Both type 1 and type 2 diabetes originate from autoimmune destruction of the pancreatic beta cells. The cause is unknown. Type 1 diabetes makes up 5%−10% of all diabetes cases. There are two subdivisions of type 1 diabetes: type 1A (*immune-mediated*), and type 1B (*idiopathic* or *nonimmune-related*). In the United States as well as in Europe, nearly all type 1 diabetics (90%−95%) have type 1A immune-mediated diabetes.

Visit https://ebookmass.com today to explore

a vast collection of ebooks across various genres, available in popular formats like PDF, EPUB, and MOBI, fully compatible with all devices. Enjoy a seamless reading experience and effortlessly download high-quality materials in just a few simple steps. Plus, don’t miss out on exciting offers that let you access a wealth of knowledge at the best prices!

FIGURE 1.2 Painting of Avicenna.

Type 2 diabetes was previously described as *non-insulin-dependent diabetes*. Hyperglycemia accompanies a relative insulin deficiency, not a total lack of insulin. Insulin therapy may be required to achieve glycemic control. Type 2 diabetes makes up 90%−95% of all diabetes cases. The majority of people with this type are of older age, and are overweight. Today, more adolescents and children are being diagnosed with type 2 diabetes than ever before.

Other specific types of diabetes were previously described as conditions of *secondary diabetes*. In these types, there is a link between the diabetic condition and various other syndromes or conditions. These include disease of the pancreas, removal of tissue from the pancreas, **pheochromocytoma, Cushing syndrome**, or **acromegaly**. For example, Cushing syndrome or acromegaly may cause *diabetogenic* effects of excess hormone levels. When one of these endocrine disorders causes hyperglycemia, it is because of increased production of glucose in the liver, or from decreased use of glucose by the cells.

Other causes of secondary diabetes include single gene defects of beta cell function, which usually develop before 25 years of age. Genetic defects of beta cell function cause dysregulation of insulin secretion because of a defect in generation of **glucokinase**. Genetic defects in insulin action cause pediatric syndromes with mutations of the insulin receptors. The most common complication of **cystic fibrosis** is secondary diabetes, especially in patients under 10 years of age. *Cystic fibrosis-related diabetes* is believed to be caused by insulin deficiency related to scarring of the pancreas. This causes loss or destruction of the insulin-producing beta cells. Blood glucose can also be elevated by diuretics, or after an organ or tissue transplant.

The final type is called *gestational diabetes*, which develops during pregnancy. It is caused by a combination of insulin resistance with impaired secretion of insulin. Today, this type is increasing, and occurs in 3%−7% of pregnancies in the United States. Intensity of hyperglycemia is extremely varied. Risk factors for gestational diabetes include significant family history of type 2 diabetes, *glycosuria*, **polycystic ovary disease**, extreme obesity, prior history of

gestational diabetes, and prior delivery of an infant that was large for gestational age. A pregnant woman must have risk assessment for diabetes during their first prenatal visit. Women with gestational diabetes are at higher risk for many complications.

SIGNS AND SYMPTOMS

Diabetes mellitus can develop quickly, or following symptoms that are vague and do not point to any specific disease or condition, which is called **insidious onset**. For type 1 diabetes, signs and symptoms usually develop quickly. For type 2 diabetes, they are more insidious. Often, the presence of the disease is discovered during routine medical check-ups, or when a patient requires medical treatment for other conditions.

There are three common signs and symptoms of diabetes, all of which are closely related to glycosuria and hyperglycemia. They are as follows:

- **Polydipsia**—excessive thirst; this is caused by intracellular dehydration, which follows increasing blood glucose levels and water being removed from body cells as well as from the hypothalamic thirst center. Polydipsia is an early symptom of type 2 diabetes that is often not noticed, especially in patients whose blood glucose levels have gradually been increasing.
- **Polyphagia**—excessive hunger; this is usually a component of type 1 diabetes, not type 2 diabetes. It usually occurs because of starvation of the cells as well as depletion of cellular proteins, fats, and carbohydrates.
- **Polyuria**—excessive urination; since glucose is a small and osmotically active molecule, elevated blood glucose levels result in the amounts that can be filtered by the glomeruli of the kidneys to exceed the amount able to be reabsorbed by the kidney tubules. The result is glycosuria and significant losses of water via the urine.

In uncontrolled type 1 diabetes, though the appetite may be normal or increased, weight loss commonly occurs. **Osmotic diuresis** causes loss of body fluids. Ketoacidosis causes fluid loss, which may be worsened by vomiting. Body tissue is also lost, since insufficient insulin causes the body to use both cellular proteins and stored fat for energy. Weight loss is often very different between type 1 and type 2 diabetes. With uncomplicated type 2 diabetes, patients are usually obese.

Hyperglycemia causes a variety of other symptoms. These include fatigue, chronic blurred vision, skin infections, and paresthesias. Fatigue and weakness occur due to lowered plasma volume. Blurred vision is linked to exposure of the lens and retina of the eye to fluids that are **hyperosmolar**. Chronic skin infections are more common in type 2 diabetes. Paresthesias are caused by temporary dysfunction of peripheral sensory nerves. The type 2 diabetic often first seeks medical treatment when these symptoms develop. Both glycosuria and hyperglycemia increase the likelihood of growing yeast organisms in the body. In diabetic women, there is often vulvovaginitis and itching (pruritus) due to *Candida* infections. These are commonly the initial complaints made by affected women. In men, secondary *Balanitis* may develop from a *Candida* infection.

PATHOPHYSIOLOGY

In type 1 diabetes, the immune system attacks the pancreatic beta cells, destroying them, and stopping insulin production. Deficiency of the beta cells leads to total insulin deficiency. This autoimmune disease involves **antiinsulin** or *anti-islet cell antibodies* to become present in the blood. This is followed by infiltration of lymphocytes and pancreatic islet destruction. While these processes take some time to develop, type 1 diabetes symptoms can manifest over only a few days to a few weeks. Additional autoimmune conditions linked to type 1 diabetes include **hypothyroidism** and **vitiligo**. Insulin therapy is always required for type 1 diabetes, which does not respond to oral drugs that stimulate insulin release.

Type 2 diabetes develops when the body becomes unable to produce enough insulin to meet its requirements. Beta cell deficiency exists, along with **peripheral insulin resistance**, which is defined as insulin levels in the blood being high while there is no **hypoglycemia**. It may be caused by variances in insulin receptors that regulate the effects of insulin. The main cause of insulin resistance is obesity. Over time, most type 2 diabetics must take insulin when oral drugs are unable to stimulate sufficient insulin release.

Gestational diabetes results from excessive hormones that oppose insulin. During pregnancy, the mother will have high blood sugar, and be in a state of insulin resistance, possibly due to defects in the insulin receptors.

Aside from the most common manifestations of the various types of diabetes (polydipsia, polyphagia, polyuria, and weight loss), there are many other developments likely to occur (see Table 1.1).

TABLE 1.1 Additional Conditions Linked to Diabetes

Condition	Description
Diabetic foot	Caused by peripheral nerve damage and blood vessel problems because of long-term diabetes; lack of sensation and peripheral vascular disease cause lack of sensation, impaired healing, and infections; small blisters, sores, or trauma are unnoticed and can quickly worsen
Diabetic ketoacidosis	Caused by type 1 diabetes from total lack of insulin and use of fatty acids for energy; uncontrolled lipid breakdown causes ketones to form, and then acidosis and ketonemia occur—**a true medical emergency**
Eye damage (diabetic retinopathy)	Caused by damage to the fine blood vessels of the retinas, due to long-term exposure to high blood glucose
Heart disease	Caused by changes in large blood vessels, which lead to atherosclerosis, cerebral artery disease, coronary artery disease, dyslipidemia, peripheral artery disease, etc.
Kidney damage	Caused by damaged to the small and large renal blood vessels; begins initially with proteinuria or increased protein outflow; may lead to end-stage renal disease
Nerve damage	May affect arms and legs (stocking-glove numbness or tingling); may affect autonomic functions, resulting in difficult digestion, erectile dysfunction, and/or impotence, gastroparesis, etc.
Nonketotic hyperosmolarity	Caused by an extreme increase of blood sugar (in type 2); though insulin still suppresses synthesis of ketones, high blood sugar results in excessive concentration or osmolarity of blood; this causes dieresis, collapse of blood vessels, and cardiovascular shock—**a true medical emergency**
Poor wound healing; gum and other infections	Caused by increased blood glucose, which is a good source of nutrition for microbes, and due to diminished immunity

DIAGNOSIS

Though the diagnosis of diabetes may have originated by the actual tasting of urine for sweetness, there have been many developments in diabetes diagnosis over centuries. The first was the boiling of diabetic urine, by Paracelsus, which revealed approximately four ounces of salt. In 1776, Matthew Dobson did a similar experiment involving boiling of urine, but this revealed a substance that looked and tasted like brown sugar. John Rollo recorded the foods eaten by his diabetic patients, and their quantities. He then evaporated their urine and weighed the cake-like residue, observing that carbohydrates in the diet increased sugar levels, while eating animal products reduced it. This led to him suggesting appropriate dietary modifications. The first clinical tests for glycosuria were developed by Karl Trommer in 1841. Urine samples were treated with a strong acid that caused hydrolysis of disaccharides into monosaccharides. He then neutralized the solution and added a copper sulfite solution, followed by an excess of alkali. This was boiled and then analyzed to see if a brick-red colored *cuprous oxide* precipitate was formed, which indicated the presence of glucose. Frederick Pavy used ammonia in an experiment that resulted in the production of the original urinalysis tablets.

After 1900, tests were simplified and improved to determine urine and blood glucose levels. In 1941, the first "stick" or "strip" tests, known as "Clinitest" were developed, which were based on copper sulfate reduction. This was followed by the more accurate "Clinistix" test, based on enzymatic reaction of glucose oxidase. More recent diagnostic developments have centered on measuring blood glucose levels and recording how patients respond to intake of oral glucose. The *oral glucose tolerance test* (OGTT) was developed in 1979, in which the patient swallows 75 g of glucose. The blood glucose levels are measured 2 hours later. This established the criterion to diagnose diabetes: fasting blood glucose of 7.8 mmol/L or higher; or a 2-hour OGTT value of 11.1 mmol/L or higher. In 1997, the diagnosis of diabetes was updated as follows:

- A fasting blood glucose of 7.0 mmol/L or higher (with polydipsia, polyuria, and weight loss)
- A random blood glucose of 11.1 mmol/L or higher
- A 2-hour OGTT value of 11.1 mmol/L or higher

The detailed diagnosis of various types of diabetes will be discussed in Chapter 10, Diagnosis.

PREVALENCE OF DIABETES IN THE UNITED STATES

According to the Centers for Disease Control and Prevention (CDC), there has been a large increase in the amount of diagnosed cases of diabetes in the United States. The statistics began being collected in 1958, and have continued until 2015. In 1958, 0.93% of the population was diagnosed with diabetes, and this numbered as 1.58 million people. By 2015, the percentage had reached 7.4% and the actual amount of people affected was 23.35 million. However, today there are over 30 million. Table 1.2 shows these statistics on diabetes prevalence in the United States.

The CDC has also created a graph showing the same information but in a more visual format (see Fig. 1.3).

TABLE 1.2 Percentage and Number of US Population with Diagnosed Diabetes

Year	Percentage	Number (in millions)
1958	0.93	1.58
1959	0.87	1.49
1960	0.91	1.59
1961	1.05	1.87
1962	1.06	1.91
1963	1.15	2.10
1964	1.24	2.31
1965	1.27	2.39
1966	1.45	2.77
1967	1.61	3.09
1968	1.62	3.18
1969	N/A	N/A
1970	N/A	N/A
1971	N/A	N/A
1972	N/A	N/A
1973	2.04	4.19
1974	N/A	N/A
1975	2.29	4.78
1976	2.36	4.97
1977	N/A	N/A
1978	2.37	5.19
1979	2.49	5.47
1980	2.54	5.53
1981	2.51	5.65
1982	2.52	5.73
1983	2.45	5.61
1984	2.59	6.00
1985	2.62	6.13
1986	2.78	6.56
1987	2.77	6.61

(*Continued*)

TABLE 1.2 (Continued)

Year	Percentage	Number (in millions)
1988	2.56	6.16
1989	2.66	6.47
1990	2.52	6.21
1991	2.90	7.21
1992	2.93	7.37
1993	3.06	7.78
1994	2.98	7.74
1995	3.30	8.66
1996	2.89	7.63
1997	3.80	10.11
1998	3.90	10.48
1999	4.00	10.87
2000	4.40	12.05
2001	4.75	13.11
2002	4.84	13.49
2003	4.93	14.10
2004	5.29	15.24
2005	5.61	16.32
2006	5.90	17.32
2007	5.86	17.40
2008	6.29	18.81
2009	6.86	20.67
2010	6.95	21.13
2011	6.78	20.74
2012	6.96	21.47
2013	7.18	22.30
2014	7.02	21.95
2015	7.40	23.35

N/A = not available.
http://www.cdc.gov/diabetes/data.

TREATMENT

Perhaps the first actual "treatment" for diabetes was by the Persian physician Avicenna (980–1037). He used a mixture of the flowering plant called *lupine*, the herb called *trigonella* (specifically, *fenugreek*), and the herb called **zedoary seed** to produce a significant reduction in excretion of sugar. This treatment is still used in parts of the world today.

In 1921, the most significant development in treating diabetes occurred in Canada, when Frederick Banting (Fig. 1.4) and Charles Best successfully isolated the hormone *insulin*. Within just 1 year, insulin treatments began to be given to diabetics, saving many lives. These scientists had correctly reasoned that diabetes was due to a malfunction of the pancreas. With the help of J.J.R. Macleod and J.B. Collip, they were able to purify insulin so that it could be administered to diabetics. Even patients who were in diabetic comas were able to be treated successfully.

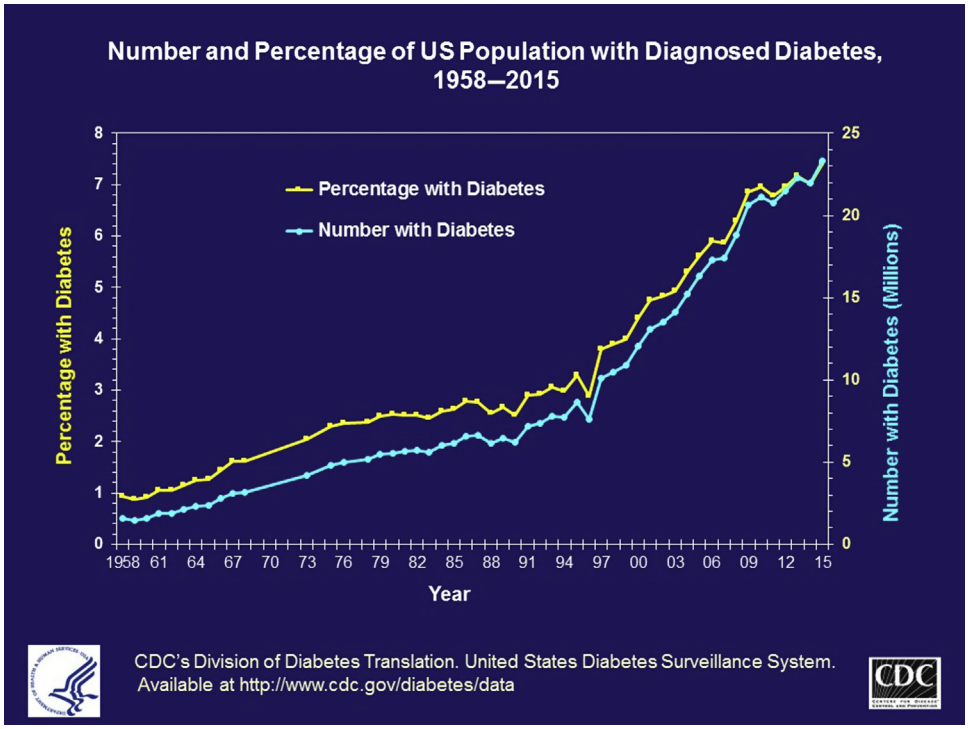

Number and Percentage of US Population with Diagnosed Diabetes, 1958–2015
Percentage with Diabetes
Number with Diabetes
Percentage with Diabetes
Number with Diabetes (Millions)
Year
1958 61 64 67 70 73 76 79 82 85 88 91 94 97 00 03 06 09 12 15
0 1 2 3 4 5 6 7 8
0 5 10 15 20 25
CDC's Division of Diabetes Translation. United States Diabetes Surveillance System.
Available at http://www.cdc.gov/diabetes/data
CDC

FIGURE 1.3 Graph of diagnosed diabetes in the United States over time.

FIGURE 1.4 Photo of Frederick Banting.

In 1942, the chemist named Marcel Janbon and his team were studying sulfonamide antibiotics, when they discovered that the compound called *sulfonylurea* induced hypoglycemia. It did this by stimulating the pancreatic cells to release insulin. In 1982, a replicated version of human insulin was developed and marketed as Humulin N. It was a long-acting form of insulin that was different than natural forms. The drug was injected just under the skin, once or twice per day. In 1984, the oral medication called Glucotrol was approved to treat type 2 diabetes. In 1995, the oral medication called Glucophage was approved for type 2 diabetes. Since then, a large variety of different medications have been successfully used to manage the various types of diabetes. A more detailed discussion of treatments of diabetes is found in Chapter 11, Treatment of Diabetes.

LESSONS FROM THE HISTORY OF DIABETES

Since diabetes was first identified in India during the 5th century BC, there have been many discoveries and facts uncovered about the disease. The disease was named because early scientists actually tasted diabetic urine and discovered it to be sweet. Greek physicians found that exercise of various types helped to alleviate the excessive urination caused by the disease. The name "diabetes" was given to the disease, which is a Greek word meaning "siphon," since diabetic people urinated so often. Many cultures were aware of the factors involved in diabetes, but it was not until the 18th century that a differentiation was made between diabetes mellitus and diabetes insipidus. In 1889, the role of pancreas was realized, in relation to development of diabetes.

In 1910, Sir Edward Albert Sharpey-Schafer discovered that diabetes was caused by a lack or insufficiency of insulin. The name "insulin" came from the Latin word "insula," which meant *island*, and was a reference to the *islets of Langerhans* in the pancreas, which produce insulin. In 1919, Frederick Allen introduced a therapy of strict dieting or starvation treatment, which successfully managed diabetes. The isolating of insulin in 1921 resulted in the scientists involved receiving the Nobel Prize in Physiology or Medicine. They made their patent free of charge so that millions of diabetics could have easy access to insulin. In 1936, Sir Harold Percival Himsworth differentiated type 1 and type 2 diabetes as being clinically unique. In 1940, the American Diabetes Association was founded to address increasing rates of diabetes and its related complications. In the 1950s, about one in five type 1 diabetics died within 20 years of diagnosis, and one in three died within 25 years. We have learned so much that today, only about 3.5% of type 1 diabetics die within 20 years of diagnosis, and only 7% die within 25 years. As late as 1988, metabolic syndrome, which is partially formed by diabetes mellitus, was discovered by Gerald Reaven.

Over history, we have learned that diabetes is a complex disorder of a heterogeneous nature. We know that insulin resistance is essential in the pathogenesis of type 2 diabetes. Metabolic syndrome is a clinical phenotype that includes insulin resistance, upper body obesity, hypertriglyceridemia, low levels of high-density lipoprotein cholesterol, and hypertension. It identifies people who are at high risk for glucose intolerance and diabetes, as well as cardiovascular disease. Genetic studies have identified more than 40 genetic variants that increase the risk for type 2 diabetes.

FURTHER READING

[1] Ali H, Anwar M, Ahmed T, Chand N. Diabetes mellitus from antiquity to present scenario and contribution of Greco-Arab physicians. JISHIM 2006;5:46–50.

[2] American Diabetes Association. *Diabetes Statistics*, 2013. http://www.diabetes.org/diabetes-basics/diabetes-statistics/.

[3] American Diabetes Association. Diabetes Symptoms, 2015. http://www.diabetes.org/diabetes-basics/symptoms/.

[4] American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2013;36(Suppl. 1):S62–9.

[5] American Diabetes Association. Standards of medical care in diabetes. Diabetes Care 2010;33:S11–61.

[6] Umpierrez GE. Cardiovascular outcomes of treatments available for patients with type 1 and 2 diabetes. An issue of endocrinology and metabolism clinics of North America. Elsevier; 2018.

[7] Centers for Disease Control. Division of Diabetes Translation. National Diabetes Surveillance System, 2010.

[8] Zochodne DW, Malik RA. Diabetes and the nervous system, volume 126 (handbook of clinical neurology). Elsevier; 2015.

[9] Hirsch IB. Diabetes management, an issue of medical clinics of North America (the clinics: internal medicine). Elsevier; 2014.

[10] Kirchhof M, Popat N, Malowany J. Diagnostic Review. A historical perspective of the diagnosis of diabetes, 2009. www.uwomj.com/wp-content/uploads/2013/06/v78n1.7-11.pdf.

[11] Eisenbarth GS, McCulloch DK. Pathogenesis of type 1 diabetes mellitus. UpToDate®; 2010.

[12] Harris RE. Epidemiology of chronic disease: global perspectives. Jones & Bartlett Learning, 2012.

[13] CM Porth. Essentials of pathophysiology. 4th ed. Wolters Kluwer, 2015.

[14] The History of Diabetes. Everyday health, 2009. https://www.everydayhealth.com/diabetes/understanding/diabetes-mellitus-through-time.aspx.

[15] Holtz C. Global Health Care: Issues and Policies. 3rd ed. Jones & Bartlett Learning; 2016.

[16] Hall JE. Guyton and Hall's medical physiology. 12th ed Elsevier Saunders; 2011. p. 939–54.
[17] Porter RS, Kaplan JL. The merck manual of diagnosis and therapy. 19th ed Merck Sharp & Dohme Corp; 2011.
[18] The past 200 years in diabetes. N Engl J Med 2018. www.nejm.org/doi/10.1056/nejmra1110560.
[19] News Medical Life Sciences. Diabetes Pathophysiology, 2016. https://www.news-medical.net/health/diabetes-pathophysiology.aspx.
[20] News Medical Life Sciences. History of Diabetes, 2017. https://www.news-medical.net/health/history-of-diabetes.aspx.
[21] NIH Research Portfolio Online Reporting Tools (RePORT). Diabetes, Type 1, 2013. https://report.nih.gov/nihfactsheets/viewfactsheet.aspx?csid = 120&key = d.
[22] Daneman D. Pediatric Endocrinology and Diabetes, An Issue of Pediatric Clinics of North America. Elsevier; 2015.
[23] Heidelbaugh JJ. Type II Diabetes Mellitus: A Multidisciplinary Approach (Clinics Collections). Elsevier; 2014.
[24] Verbeeten KC, Elks CE, Daneman D, Ong KK. Association between childhood obesity and subsequent Type 1 diabetes: a systematic review and meta-analysis. Diabet Med 2011;28(1):10–18.
[25] World History Project. *Diabetes treatment history timeline*, 2018. https://worldhistoryproject.org/topics/diabetes-treatment-history.

Chapter 2

The Pancreas and Classifications of Insulin

In adults, the **pancreas** is oriented transversely and extends from the C-loop of the duodenum to the hilum of the spleen. Though its name is derived from a Greek word, *pancreas*, which means *all flesh*, the pancreas is actually a complicated organ with many lobules. It has separate exocrine and endocrine components.

The *exocrine* pancreas makes up most (80%−85%) of the organ. It consists of **acinar cells** that secrete the enzymes required for digestion. Acinar cells are pyramid-shaped epithelial cells. They contain membrane-bound granules that are rich in *proenzymes*, also called **zymogens**. These include trypsinogen, chymotrypsinogen, procarboxypeptidase, **kallikreinogen**, proelastase, and prophospholipase A and B. When these proenzymes and enzymes are secreted, many ducts and ductules carry them to the duodenum, where they are activated by proteolytic cleavage in the gastrointestinal tract.

The *endocrine* pancreas is made up of approximately one million cell clusters known as the **islets of Langerhans**. These cells are scattered throughout the pancreas, and secrete insulin, glucagon, and somatostatin. The islet cells make up only 1%−2% of the pancreas.

THE PANCREAS

The pancreas is located below and behind the stomach, but in front of the spine. It is a spongy gland of 12−15 cm (6−10 in.) in length, and is mostly **retroperitoneal**. The pancreas is grayish-pink in color, and weighs about 60 g. The head of the pancreas is on the right side of the abdomen. It is connected to the first section of the small intestine, the *duodenum*, via the small *pancreatic duct*. The narrow *tail* of the pancreas extends to the left side of the body (see Fig. 2.1A). The pancreas is surrounded by the small intestine, stomach, liver, and spleen. Because of the deep location of the pancreas, when tumors develop, they are rarely able to be *palpated* by pressing on the abdomen.

The Pancreatic Islets

The tissues of the pancreas contain approximately one to two million clusters of cells that are called **pancreatic islets** or *islets of Langerhans* (see Fig. 2.1B). The pancreatic islets actually make up <2% of the total pancreatic tissues. However, they are extremely important because they secrete hormones needed primarily in the regulation of **glycemia**, which is the blood glucose concentration. Each islet is about 75 × 175 μm in size, and contains as little as a few cells to approximately 3000 cells. These cells are of three primary types: the alpha cells, which make up 20% of the total amounts of cells; the beta cells, 70%; and the delta cells, 5%. All of the islet cells directly respond to nutrient levels in the blood that are related to eating and fasting. Approximately 5% of the pancreatic cells consist of pancreatic polypeptide (PP) cells. There are four types of hormone-secreting cells:

- **Alpha cells**—which secrete glucagon
- **Beta cells**—which secrete insulin and amylin
- **Delta cells**—which secrete gastrin and somatostatin
- **F (or PP) cells**—which secrete PP; this stimulates gastric secretions, and antagonizes cholecystokinin.

These hormones regulate the metabolism of carbohydrates, fats, and proteins.

The pancreatic islets are innervated by nerves from the sympathetic and parasympathetic divisions of the autonomic nervous system. The *vagus nerves* innervate the pancreas, liver, gallbladder, stomach, small intestine, and the proximal half of the large intestine.

Epidemiology of Diabetes. DOI: https://doi.org/10.1016/B978-0-12-816864-6.00002-X
© 2019 Elsevier Inc. All rights reserved.

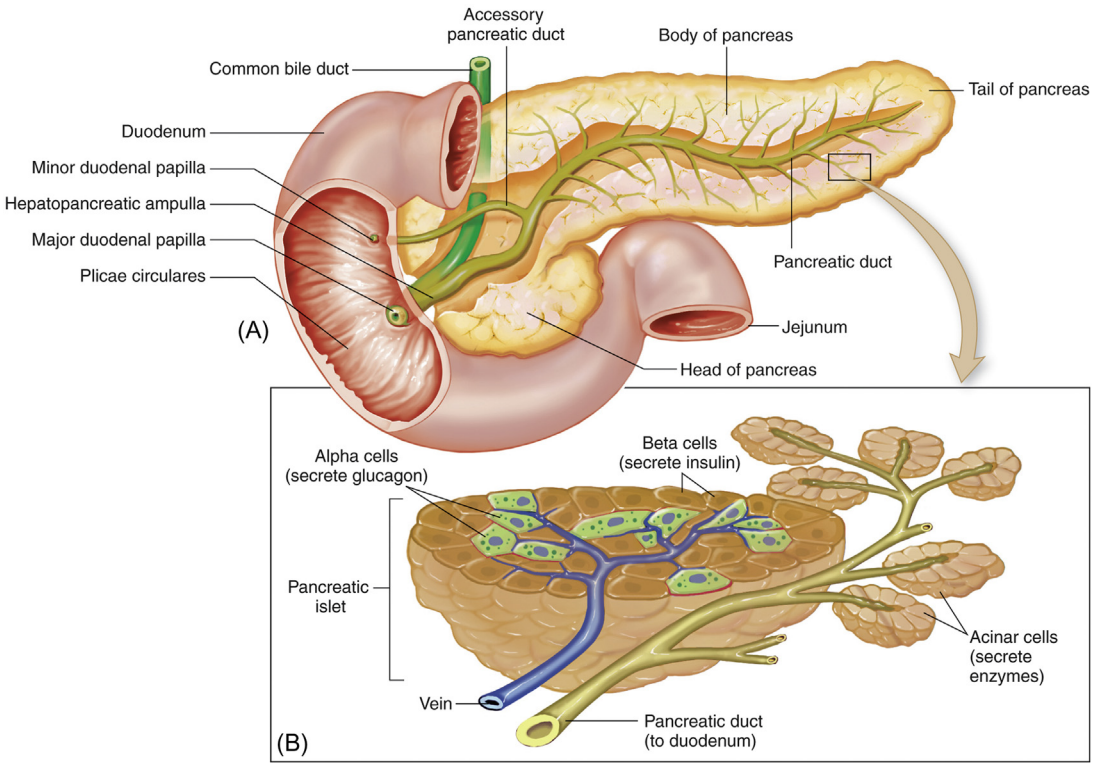


FIGURE 2.1 The pancreas. (A) Pancreas dissected to show main and accessory ducts. The main duct may join the common bile duct, as shown here, to enter the duodenum by a single opening at the major duodenal papilla, or the two ducts may have separate openings. The accessory pancreatic duct is usually present and has a separate opening into the duodenum. (B) Exocrine glandular cells (around small pancreatic ducts) and endocrine glandular cells of the pancreatic islets (adjacent to blood capillaries). Exocrine pancreatic cells secrete pancreatic juice, alpha endocrine cells secrete glucagon, and beta cells secrete insulin. *Adapted from K.T. Patton, G.A. Thibodeau.* The Human Body in Health & Disease*, 7 ed., St Louis, 2018, Elsevier.*

The alpha, beta, and delta cells are most numerous in the anterior lobe of the pancreas. Perfusion of this lobe comes from branches of the **superior mesenteric artery**. This is a large and unpaired artery arising from the abdominal aorta at the L1 level, just below the **celiac trunk**. It runs deep to the pancreas, entering the mesentery, where its many anastomosing branches serve almost all of the small intestine. Branches of the celiac trunk perfuse the posterior lobe of the pancreas. The **common hepatic artery** branches to the pancreas, stomach, and duodenum. The **splenic artery** branches to the pancreas and stomach, with its branches terminating in the spleen. Although the pancreatic islets make up only about 1% of the mass of the pancreas, they receive 10% of pancreatic blood flow. This is essential so that islet hormones can be oxygenated and delivered to target cells. The **splenic vein** collects blood from parts of the pancreas, stomach, and spleen. It joins the superior mesenteric vein, forming the hepatic portal vein.

Functions of the Pancreas

The functions of the pancreas are primarily as an exocrine digestive gland. It is essential in converting food into fuel for the body's cells. When healthy, the pancreas produces specific chemicals in exact quantities, when they are needed, to digest foods. Its exocrine glands produce digestive enzymes that include *trypsin* and *chymotrypsin* to digest proteins. It also produces *amylase* to digest carbohydrates and *lipase* to break down fatty acids and cholesterol. As food enters the stomach, the pancreatic juices are released into a duct system ending in the main *pancreatic duct*. The pancreatic duct joins the *common bile duct* to form the **ampulla of Vater** at the duodenum. The common bile duct actually originates in the liver and gallbladder. It produces *bile*, another important digestive juice. Therefore, bile and pancreatic juices are released into the duodenum to help digest proteins, carbohydrates, and fats. A healthy pancreas produces about 2.2 pints (1 L) of enzymes each day. The endocrine functions of the pancreas occur via the pancreatic islets, primarily via the hormones, *insulin* and *glucagon*. The maintenance of blood glucose levels is crucial for normal body functions.

Visit https://ebookmass.com today to explore

a vast collection of ebooks across various genres, available in popular formats like PDF, EPUB, and MOBI, fully compatible with all devices. Enjoy a seamless reading experience and effortlessly download high-quality materials in just a few simple steps. Plus, don’t miss out on exciting offers that let you access a wealth of knowledge at the best prices!

Exploring the Variety of Random Documents with Different Content

Marplot
Match'em
Meddler
Mendall
Merlin
Messmate
Monarch
Motley
Mounter
Mungo

M. *bitches*.

Madcap
Magic
Minion
Mischief
Music

N. *dogs*.

Nestor
Nettler
Newsman
Nimrod
Noble
Nonsuch

N. *bitches*.

Nimble
Noisy
Novice

P. *dogs*.

Pealer

Perfect
Phœbus
Pilgrim
Pilot
Piper
Playful
Prattler
Presto
Primate
Prowler
Prosper

P. *bitches*.

Patience
Placid
Pliant
Precious
Priestess
Prudence

R. *dogs*.

Racer
Rambler
Random
Ranger
Ransack
Ranter
Rattler
Rector
Render
Rifler
Ringwood
Risker
Rockwood
Rouser

Rover
Rumbler
Rusher

R. *bitches*.

Racket
Rapid
Rattle
Ruin
Rummage

S. *dogs*.

Samson
Saunter
Scalper
Scamper
Scourer
Scramble
Scuffler
Sharper
Shifter
Singer
Skirmish
Smoker
Songster
Soundwell
Spanker
Spinner
Spoiler
Sportsman
Squabbler
Squeaker
Steady
Stickler
Stringer

Stringer
Stripling
Striver
Stroker
Struggler
Sturdy
Sylvan

S. *bitches*.

Sappho
Skilful
Speedy
Spitfire
Sportful
Sprightly
Stately
Strumpet
Sybil

T. *dogs*.

Tackler
Tamer
Tangent
Tartar
Tattler
Taunter
Teaser
Terror
Thrasher
Thumper
Thwacker
Tickler
Tomboy
Topper
Torrent

Touchstone
Trampler
Trimbush
Trimmer
Trojan
Trouncer
Truant
Trueboy
Trueman
Trusty
Tryall
Tuner
Twig’em

T. *bitches.*

Tattle
Telltale
Tempest
Termagant
Testy
Trifle
Trollop
Truelass
Tuneful

V. *dogs.*

Vagrant
Valid
Vaulter
Venture
Vexer
Victor
Vigorous
Viper
Volant

Voucher

V. *bitches.*

Vanquish
Venomous
Vicious
Vivid
Vixen
Vocal

W. *dogs.*

Warbler
Warrior
Wayward
Wellbred
Whynot
Wildair
Wildman
Wilful
Woodman
Workman
Wrangler
Wrestler

W. *bitches.*

Waggery
Waggish
Wagtail
Wanton
Warfare
Warlike
Waspish
Watchful
Welcome

Whimsey
Wildfire
Wishful
Worry
Wrathful.

Brown—Whitaker—Beckford, &c.

HOUND, *v.* To set on the chase; to hunt, to pursue.

HOUR, *s.* The twenty-fourth part of a natural day; the space of sixty minutes.

HOURGLASS, *s.* A glass filled with sand, which, running through a narrow hole, marks the time.

HOUSE, *v.* To harbour, to admit to residence; to shelter, to keep under a roof; to take shelter, to keep the abode.

HOUSEDOG, *s.* A mastiff kept to guard the house.

HOUSING, *s.* Cloth originally used to keep off dirt, now added to saddles as ornamental.

HOWL, *v.* To cry as a wolf or dog; to utter cries in distress.

HOWL, *s.* The cry of a wolf or dog; the cry of a human being in horror.

HOY, *s.* A large boat, sometimes with one deck.

HUCHO, *s.* A fish of the genus Salmo.

The hucho is the most predatory fish of the salmo genus, and is made like an ill-fed trout, but longer and thicker. He has larger teeth, more spines in the pectoral fin, a thicker skin, a silvery belly, and dark spots only on the back and sides. I have never seen any on the fins. The ratio of his length to the girth is as eight to eighteen, or, in well-fed fish, as nine to twenty; and a fish, eighteen inches long by eight in girth, weighed 16,215 grains. Another, two feet long, eleven inches in girth, and three inches thick, weighed 4lbs. 2¼ oz. Another, twenty-six inches long, weighed 5lbs. 5oz. Of the spines in the fins, the anal has nine, the caudal twenty, the ventral nine, the dorsal twelve, the pectoral seventeen: having numbered the spines in many, I give this as correct. The fleshy fin belonging to the genus is, I think, larger in this species than in any I have seen. Bloch, in his work on fishes, states, that there are black spots on all the fins, with the exception of the anal, as a character of this fish: and professor Wagner informs me, he has seen huchoes with this peculiarity; but, as I said before, I never saw any fish with spotted fins; yet, I have examined those of the Danube, Save, Drave, Mur, and Izar: perhaps, this is peculiar to some stream in Bavaria; yet the huchoes in the collection at Munich have it not. The hucho is found in most rivers tributary to the Danube—in the Save and Laybach rivers always; yet

the general opinion is, that they run from the Danube twice a year, in spring and autumn. I can answer for their migration in spring, having caught several in April, in streams connected with the Save and Laybach rivers, which had evidently come from the still dead water into the clear running streams, for they had the winter leech, or louse, of the trout upon them; and I have seen them of all sizes in April in the market at Laybach, from six inches to two feet long; but they are found much larger, and reach thirty, or even forty pounds. It is the opinion of some naturalists that it is only a fresh-water fish; yet this I doubt, because it is never found beyond certain falls—as in the Traun, the Drave, and the Save; and, there can be no doubt, comes into these rivers from the Danube; and probably in its largest state, is a fish of the Black Sea. Yet it can winter in fresh water; and does not seem, like the salmon, obliged to haunt the sea, but falls back into the warmer waters of the great rivers; from which it migrates in spring, to seek a cooler temperature, and to breed. The fishermen at Gratz say they spawn in the Mur, between March and May. In those I have caught at Laybach, which, however, were small ones, the ova were not sufficiently developed to admit of their spawning that spring. Marsigli says that they spawn in the Danube in June. You have seen how violently they pursue their prey; I have never taken one without fish in his stomach; yet, when small, they will take a fly. In the Kleingraben, which is a feeder to the Laybach river, and where they are found of all sizes—from twenty pounds downwards—the little ones take a fly, but the large ones are too ravenous to care about so insignificant a morsel, and prey like the largest trout, often hunting in company, and chasing the small fish into the narrow and shallow streams, and then devouring them.

The hucho, as you have seen, preys with great violence, and pursues his object as a foxhound or a greyhound does. I have seen him in repose; they lie like pikes, perfectly still, and I have watched one for many minutes, that never moved at all. In this respect their habits resemble those of most carnivorous and predatory animals. It is probably in consequence of these habits, that they are so much infested by lice, or leeches, which I have seen so numerous in spring

as almost to fill their gills, and interfere with their respiration, in which case they seek the most rapid and turbulent streams to free themselves from these enemies. They are very shy, and, after being hooked, avoid the baited line. I once saw the hucho, for which I was fishing, follow the small fish, and then the lead of the tackle; it seemed as if this had fixed his attention, and he never offered at the bait afterwards. I think a hucho that has been pricked by the hook becomes particularly cautious, and possesses, in this respect, the same character as the salmon. In summer, when they are found in the roughest and most violent currents, their fins (particularly the caudal fin) often appear worn and broken; at this season they are usually in constant motion against the stream, and are stopped by no cataract or dam, unless it be many feet in height, and quite inaccessible. In the middle of September, I have caught huchoes perfectly clean in rapid cool streams, tributary to the Laybach and the Save rivers; and, from the small development of their generative system at this time, I have no doubt that they spawn in spring. On the 13th of September, 1828, I caught, by spinning the small dead fish, three huchoes that had not a single leech upon their bodies, and they were the first fish of the kind I ever saw free from these parasites.

They migrate generally when the water is foul, and, except in the spring and autumn, do not so readily run at the bait. I was once nearly a month seeking for one in rivers in which they are found, between the end of June and that of July, without being able to succeed in even seeing one alive; and, as far as my information goes, the two places where there is most probability of taking them, are at Laybach and Ratisbon, in the tributary streams to the Save, and in the Danube; and the best time, in the first of these situations, is in March and April, and, in the second, in May. I am told, likewise, that the Izar, which runs by Munich, is a stream where they may be caught, when the water is clear: and I have seen in the fish market at Munich very large huchoes.

I am inclined to believe that the hucho is to be found in some of the mountain loughs in Connaught. Certainly I have seen fish of the salmo genus, taken in rivers communicating with deep lakes in the hills, which strikingly resemble the fish described by Sir Humphry.—*Salmonia—Editor.*

HUE, *s.* Colour, dye; a clamour, a legal pursuit.

HULK, *s.* The body of a ship; anything bulky and unwieldy.

HUMBLEBEE, *s.* A buzzing wild bee; an herb.

HUMOUR, *s.* Moisture; the different kinds of moisture in man's body. *Humours of the eye* are these—the *aqueous or watery*, which lies in the forepart of the globe; the *crystalline*, next to the *aqueous*; and the *vitreous or glassy* humour, which is larger than the rest, and fills the backward cavity of the eye.—*Crabbe.*

HUNT, *v.* To chase wild animals; to pursue, to follow close; to search for; to direct or manage hounds in the chase.

Hunting among the Britons is of great antiquity. Dio Nicæus, speaking of the inhabitants of the northern parts of this island, tells us, they were a fierce and barbarous people, who tilled no ground, but lived upon the depredations they committed in the southern districts, or upon the food they procured by hunting. Strabo also says, that the dogs bred in Britain were highly esteemed upon the continent, on account of their excellent qualities or hunting; and these qualities, he seems to hint, were natural to them, and not the effect of tutorage by their foreign masters.

After the expulsion of the Danes, and during the short restoration of the Saxon monarchy, the sports of the field still maintained their ground. Edward the Confessor, whose disposition seems rather to have been suited to the cloister than to the throne, would join in no other secular amusements; but he took the greatest delight, says William of Malmsbury, "to follow a pack of swift hounds in pursuit of game, and to cheer them with his voice."

During the tyrannical government of William the Norman, and his two sons who succeeded him, the restrictions concerning the killing of game were increased. The privilege of hunting in the royal forests was confined to the king and his favourites; and, to render these receptacles for the beasts of the chase more capacious, or to make new ones, whole villages were depopulated, and places of divine worship overthrown.

King John was particularly attached to the sports of the field; and his partiality for fine horses, hounds, and hawks, is evident, from his frequently receiving such animals, by way of payment, instead of money, for the renewal of grants, fines, and forfeitures, belonging to the crown.

Edward III. took so much delight in hunting, that even at the time he was engaged in war with France, and resident in that country, he had with him in his army sixty couple of stag hounds, and as many hare hounds, and every day he amused himself with hunting or hawking.

James I. preferred the amusement of hunting to hawking or shooting. It is said of this monarch, that he divided his time betwixt his standish, his bottle, and his hunting; the last had his fair weather, the two former his dull and cloudy.

It would be a needless task, to quote the passages in the poetical and prose writings of the last three centuries, which prove that this favourite pastime has lost nothing of its relish in modern times, but, on the contrary, seems to be more generally practised.

If you have the whole country to yourself, and can hunt on either side of your house, as you please, never (when you can help it) fix your place of hunting till you see what the weather is.

Before a huntsman goes into the kennel to draft his hounds, let him determine within himself the number of hounds he intends to take out; as likewise the number of young hounds that he can venture in the country where he is going to hunt. Different countries may require different hounds: some may require more hounds than others. It is not an easy matter to draft hounds properly; nor can any expedition be made in it without some method.

When the place of meeting and time are fixed, every huntsman ought to be as exact to them as it is possible for him to be. On no account is he to be before the time; yet, on some occasions, it might be better, perhaps, for the diversion, were he permitted to be after it. The course your huntsman intends to take in drawing ought always to be well understood before he leaves the kennel.

If your huntsman, without inconvenience, can begin drawing at the farthest cover down the wind, and so draw from cover to cover up the wind till you find, let him do it.—*Vide* COVER.

While hounds are drawing for a fox, let your people place themselves in such a manner that he cannot go off unseen. I have known them lie in sheep's scrapes on the side of hills, and in small bushes, where huntsmen never think of looking for them; yet, when they hear a hound, they generally shift their quarters, and make for

closer covers. Gentlemen should take this necessary part of fox-hunting on themselves, for the whipper-in has other business to attend to.

Huntsmen, whilst their hounds are drawing, or are at fault, frequently make so much noise themselves, that they can hear nothing else: they should always have an ear to a halloo. I once saw an extraordinary instance of the want of it in my own huntsman, who was making so much noise with his hounds, which were then at fault, that a man hallooed a long while before he heard him; and when he did hear him, so little did he know whence the halloo came, that he rode a couple of miles the wrong way, and lost the fox.

Though a huntsman ought to be as silent as possible at going into a cover, he cannot be too noisy at coming out of it again; and if at any time he should turn back suddenly, let him give as much notice of it as he can to his hounds, or he will leave many of them behind him; and should he turn down the wind, he may see no more of them.

There are times when hounds should be helped, and at all times they must be kept forward: hounds will naturally tire on a cold scent, when stopped by sheep, or other impediments; and when they are no longer able to get forward, will oftentimes hunt the old scent back again, if they find that they can hunt no other. It is the judicious encouraging of hounds to hunt when they cannot run, and the preventing them from losing time by hunting too much when they might run, that distinguishes a good sportsman from a bad one. Hounds that have been well taught will cast forward to a hedge of their own accord: but you may assure yourself this excellence is never acquired by such as are left entirely to themselves.

Though I like to see fox-hounds cast wide and forward, and dislike to see them pick a cold scent through flocks of sheep to no purpose, yet I must beg leave to observe, that I dislike still more to see that unaccountable hurry which huntsmen will sometimes put themselves into the moment their hounds are at fault. Time ought always to be allowed them to make their own cast; and if a huntsman is judicious, he will take that opportunity to consider what part he himself has

next to act; but instead of this, I have seen hounds hurried away the very instant they came to a fault, a wide cast made, and the hounds at last brought back again to the very place from whence they were so abruptly taken, and where, if the huntsman had had a minute's patience, they would have hit off the scent themselves.

When your huntsman makes a cast, I hope he makes it perfect one way before he tries another, as much time is lost by going backwards and forwards. You will see huntsmen, when a forward cast does not succeed, come slowly back again: they should return as fast as they can.

In large covers, if there are many roads, in bad scenting days, when these roads are dry, or after a thaw, when they carry, it is necessary your huntsman should be near to his hounds to help them, and hold them forward. Foxes will run the roads at these times, and hounds cannot always own the scent. When they are at fault on a dry road, let not your huntsman turn back too soon; let him not stop till he can be certain that the fox is not gone on. The hounds should try on both sides the road at once: if he perceives that they try on one side only, on his return let him try the other.

If a fox runs up the wind when first found, and afterwards turns, he seldom, if ever, turns again. This observation may not only be of use to your huntsman in his cast, but may be of use to you, if you should lose the hounds.

When you are pursuing a fox over a country, the scent being bad, and the fox a long way before, without ever having been pressed, if his point should be for strong earths that are open, or for large covers, where game is in plenty, it may be acting wisely to take off the hounds at the first fault they come to; for the fox will go many miles to your one, and probably will run you out of all scent; but if he should not, you will be likely to change at the first cover you come into: when a fox has been hard pressed, it is my opinion, that he never should be given up.

A perfect knowledge of his country certainly is a great help to a huntsman: if yours, as yet, has it not, great allowance ought to be made.

In a country where there are large earths, a fox that knows the country, and tries any of them, seldom fails to try the rest. A huntsman may take advantage of this; they are certain casts, and may help him to get nearer to his fox.

Great caution is necessary when a fox runs into a village: if he is hallooed there, get forward as fast as you can. Foxes, when tired, will lie down anywhere, and are often lost by it. A wide cast is not the best to recover a tired fox with tired hounds: they should hunt him out, inch by inch, though they are ever so long about it.

A huntsman must take care, where foxes are in plenty, that he does not run the heel; for it frequently happens that hounds can run the wrong way of the scent better than they can the right, when one is up the wind, and the other down.

When a huntsman hears a halloo, and has five or six couple of hounds along with him, the pack not running, let him get forward with those which he has: when they are on the scent, the others will soon join them.

Let him lift his tail hounds, and get them forward after the rest: it can do no hurt. But let him be cautious how he lifts any hounds to get them forward before the rest: it always is dangerous, and foxes are sometimes lost by it.

When hounds are hunting a cold scent, and point towards a cover, let a whipper-in get forward to the opposite side of it. Should the fox break before the hounds reach the cover, stop them, and get them nearer to him.

When a fox persists in running in a strong cover, lies down often behind the hounds, and they are slack in hunting him, let the huntsman get into the cover to them: it may make the fox break; it may keep him off his toil; or may prevent the hounds from giving him up.

It is not often that slow huntsmen kill many foxes: they are a check upon their hounds, which seldom kill a fox but with a high scent, when it is out of their power to prevent it. Activity is the first requisite in a huntsman to a pack of fox-hounds: a want of it no judgment can make amends for; but the most difficult of all his undertakings is the distinguishing betwixt different scents, and knowing with any certainty the scent of his hunted fox. Much speculation is here required; the length of time hounds remain at fault; difference of ground; change of weather; all these contribute to increase the difficulty, and require a nicety of judgment, and a precision, much above the comprehension of most huntsmen.

When hounds are at fault, and cannot make it out of themselves, let the first cast be quick; the scent is then good, nor are the hounds likely to go over it: as the scent gets worse, the cast should be slower, and be more cautiously made. This is an essential part of hunting, and which, I am sorry to say, few huntsmen attend to. 1 wish they would remember the following rules, viz.: that, with a good scent, their cast should be quick; with a bad scent, slow; and that, when the hounds are picking along a cold scent, they are not to cast them at all.

When hounds are making a good and regular cast, trying for the scent as they go, suffer not your huntsman to say a word to them: it cannot do any good, and probably may make them go over the scent.

When hounds come to a check, a huntsman should observe the tail hounds: they are least likely to over-run the scent, and he may see by them how far they brought it. In most packs there are some hounds that will show the point of the fox, and, if attended to, will direct his cast. When such hounds follow unwillingly, he may be certain the rest of the pack are running without a scent.

Different countries require different casts: such huntsmen as have been used to a woodland and inclosed country, I have seen lose time in an open country, where wide casts are always necessary.

When you want to cast round a flock of sheep, the whipper-in ought to drive them the other way, lest they should keep running on before you.

Most huntsmen like to have all their hounds turned after them, when they make a cast: I wonder not at them for it, but I am always sorry when I see it done; for till I find a huntsman that is infallible, I shall continue to think the more my hounds spread the better: as long as they are within sight or hearing, it is sufficient. Many a time have I seen an obstinate hound hit off the scent, when an obstinate huntsman, by casting the wrong way, has done all in his power to prevent it. Two foxes I remember to have seen killed in one day by skirting hounds, whilst the huntsman was making his cast the contrary way.

When your hounds are divided into many parts, you had better go off with the first fox that breaks. The ground will soon get tainted, nor will hounds like a cover where they are often changing.

The heading a fox back at first, if the cover be not a large one, is oftentimes of service to hounds, as he will not stop, and cannot go off unseen. When a fox has been hard run, I have known it turn out otherwise; and hounds that would easily have killed him out of the cover, have left him in it.

When a fox has been often headed back on one side of a cover, and a huntsman knows there is not any body on the other side to halloo him, the first fault his hounds come to, let him cast that way, lest the fox should be gone off; and if he is in the cover, he may still recover him.

Suffer not your huntsman to take out a lame hound. If any are tender-footed, he will tell you, perhaps, that they will not mind it when they are out: probably they may not; but how will they be on the next day? A hound, not in condition to run, cannot be of much service to the pack; and taking him out at that time may occasion him a long confinement afterwards. Put it not to the trial.

All hounds go fast enough with a good scent; but it is the particular excellence of a fox-hound, when rightly managed, to get on faster with an indifferent scent than any other hound, and it is the business of a huntsman to encourage this. Every minute you lose is precious, and increases your difficulties; and while you are standing still the fox is running miles.

When hounds flag from frequent changes and a long day, it is necessary for a huntsman to animate them as much as he can: he must keep them forward, and press them on; for it is not likely, in this case, that they should over-run the scent: at these times the whole work is generally done by a few hounds, and he should keep close to them.

The many chances that are against you in fox-hunting; the changing frequently; the heading of the foxes; their being coursed by sheep-dogs; long faults; cold hunting, and the dying away of the scent; make it necessary to keep always as near to the fox as you can; which should be the first and invariable principle of fox-hunting. Long days do great hurt to a pack of fox-hounds. I set out one day last winter from the kennel at half-past seven, and returned home a quarter before eight at night, the hounds running hard the greatest part of the time. The huntsman killed one horse and tired another, and the hounds did not recover it for more than a week.

The whole system of hunting is so revolutionised that the preparation which a horse now requires is very different to what it was in former times. The hour of meeting is seldom before eleven: the find generally quick and certain; and horses are often not more than five or six hours from their stables after the best day's sport; and the ground they go over is frequently not so much as a plating race horse performs in contending three or four-mile heats. Having said this, I see no reason to doubt the propriety of feeding, sweating, and muzzling the hunter much in the same manner as the race horse, only making due and proper allowance for the relative nature of their work; particularly as to not stripping the hunter too

much of his flesh; or losing sight of the natural difference between the thorough-bred horse and the cock-tail.

There is certainly no country in the world where the sport of hunting on horseback is carried to such a height as in Great Britain at the present day, and where the pleasures of a fox-chase are so well understood, and conducted on such purely scientific principles. It is considered the *beau ideal* of hunting by those who pursue it. There can be no doubt that it is infinitely superior to stag-hunting, for the real sportsman can only enjoy that chase when the deer is sought for, and found like other game which are pursued with hounds. In the case of finding an outlying fallow-deer, which is unharboured in this manner, great sport is frequently afforded; but this is rarely to be met with in Britain. So that fox-hunting is now the chief amusement of the true British sportsman; and a noble one it is: the artifices and dexterity employed by this lively, crafty animal, to avoid the dogs, are worthy of our admiration, as he exhibits more devices for self-preservation than any other beast of the chase.

In many parts of this and the sister island, hare-hunting is much followed, but fox-hunters consider it as a sport only fit for women and old men. But although it is less arduous than that of the fox-chase, there are charms attached to it which compensate for the hard riding of the other.

The hunting match given by the Prince Esterhazy, Regent of Hungary, upon the signing the treaty of peace with France, was a day's sport, that bids fair to vie in point of blood (if the King of Naples' slaughter be excepted) with any of those recorded in modern history, as there were killed, 160 deer, 100 wild boars, 300 hares, and 80 foxes. The king had a larger extent and a longer period for the exercise of his talents, and it is proved that during his journey to Vienna, in Austria, Bohemia, and Moravia, he killed five bears, 1820 boars, 1950 deer, 1145 does, 1625 roebucks, 1121 rabbits, 13 wolves, 17 badgers, 16,354 hares, and 354 foxes; the

Welcome to our website – the perfect destination for book lovers and knowledge seekers. We believe that every book holds a new world, offering opportunities for learning, discovery, and personal growth. That’s why we are dedicated to bringing you a diverse collection of books, ranging from classic literature and specialized publications to self-development guides and children's books.

More than just a book-buying platform, we strive to be a bridge connecting you with timeless cultural and intellectual values. With an elegant, user-friendly interface and a smart search system, you can quickly find the books that best suit your interests. Additionally, our special promotions and home delivery services help you save time and fully enjoy the joy of reading.

Join us on a journey of knowledge exploration, passion nurturing, and personal growth every day!

ebookmasss.com